Central Obesity and the Metabolic Syndrome: Implications for Primary Care Providers

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Appel, S., Jones, E. & Kennedy-Malone, L. (2004). Central obesity and the metabolic syndrome: implications for primary care providers. *Journal of the American Academy of Nurse Practitioners*, *16* (8) 335-342.

Abstract:

Purpose: To describe screening measures that will determine which clients are at risk For the metabolic syndrome, common manifestations of the syndrome, preventive diagnostic considerations, and management and treatment options that primary care providers can implement.

Data Sources: Review of the clinical and research literature, supplemented with specific diagnostic criteria.

Conclusions: Central obesity is the cornerstone of the metabolic syndrome, which may lead to type 2 diabetes and cardiovascular disease. Generalized obesity is defined as body weight that is considerably greater than the ideal weight and that is distributed on all parts of the body. Generalized obesity has long been considered a significant risk factor for developing type 2 diabetes and cardiovascular disease. Those clients of ideal body weight have been considered at less risk For developing these conditions. However, this perception may not always be accurate. Weight distribution plays a major role in acquiring the metabolic syndrome. Because waist circumference is as important as overall body weight, central obesity is key to determining the risk.

Implications for Practice: The metabolic syndrome has now been given a CPT code (277.7). It is more likely that clients at risk for or with the metabolic syndrome may first be seen by a primary care provider. Primary care providers need to be able to diagnose, treat, and provide preventive interventions for the metabolic syndrome. Clients at risk will likely be identified during routine health screening. Early detection of and interventions focused on the metabolic syndrome may reduce the occurrence of type 2 diabetes and cardiovascular disease. Use of a tape measure to determine waist circumference may help the provider to identify at-risk clients who are of normal weight, and thus not previously believed to be at risk, as well as those more obviously at risk. It is necessary to determine not only patients' overall body weight but also their waist circumference. A measuring tape may be the key tool for establishing a patient's early risk for the metabolic syndrome and, ultimately, for prevention of type 2 diabetes and cardiovascular disease.

Conflict of Interest Statement: No relationship that might represent a conflict of interest exists between any of the authors and any commercial entity or product mentioned in this manuscript. No inducements have been made by any commercial entity to submit this article for publication.

Article:

DISCOVERY OF THE METABOLIC SYNDROME

In health care literature and professional seminars, the metabolic syndrome is being discussed as a mechanism for describing an array of symptoms that increase an individual's risk for the development of type 2 diabetes and cardiovascular disease *(see* Table 1). The view that generalized obesity leads to the development of these symptoms has been widely held for many years. However, mounting evidence indicates that screening For central obesity may be a more useful tool in determining the risk for the metabolic syndrome than is screening for generalized obesity as determined by body mass index (BMI; see Tables 2 and 3).

In 1947, French physician Jean Vague made the seminal observation that android, or male pattern, central obesity imposed an increased risk for cardiovascular disease (Bastard, Pieroni, & Hainque, 2000). On the basis

of his studies, it was concluded that central obesity created an environment that fostered metabolic disarray, leading to chronic diseases such as type 2 diabetes and arteriolosclerosis. Kissebah and associates in the 1970s and 1980s demonstrated that adipose cells collected from participants' abdominal area were more metabolically active than were adipose cells collected from other areas of the body. The abdominal adipose cells contributed to the development of insulin resistance (Kissebah, Adams, & Wynn, 1974; Kissebah, Alfarsi, Adams, & Wynn, 1976; Kissebah et al., 1982; Kissebah, Peiris, & Evans, 1988). The National Heart, Lung, and Blood Institute (NHLBI; 1998) defined central obesity as the presence of excess far in the abdomen **out** of proportion **to** total body fat (see Table 4). Further, the accumulation of the deep visceral abdominal adipose tissue—not only the superficial subcutaneous layer—has been associated with metabolic syndrome (Bray, 1998; Kelley, Thaete, Troost, Huwe, & Goodpaster, 2000; Smith et al., 2001).

Reaven (1988) named this link between android central obesity and the associated metabolic disarray as syndrome X. Syndrome X is now more widely referred to as the metabolic syndrome. The metabolic syndrome is defined as the presence of three or more of the following components: central obesity, hyperinsulinemia, dyslipidemia, and hypertension *(see* Table 1; Ford, Giles, & Dietz, 2002). In the early stages of the metabolic syndrome, fasting plasma glucose levels are commonly found to be normal; however, as the metabolic syndrome progresses without intervention, impaired glucose tolerance and finally type 2 diabetes may present (Ford et al.; National Cholesterol Education Program [NCEP], 2001). Although not all clients who meet the criteria for the metabolic syndrome will develop overt type 2 diabetes, all will remain at significant risk for cardiovascular disease if the syndrome persists.

Table 1 NCEP ATP III Guidelines for Diagnosis of the Metabolic Syndrome and ADA (2004) Recommendations for Fasting Glucose

Clinical Indicators of the Metabolic Syndrome	
Waist Circumference	
Men	≥ 102 cm or ≥ 40 in
Women	≥ 88 cm or ≥ 35 in
Triglycerides	≥ 150 mg/dL
HDL-C	
Men	≤ 40 mg/dL
Women	≤ 50 mg/dL
Blood Pressure	
Systolic	≥ 130 mm Hg
Diastolic	≥ 85 mm Hg
Fasting Glucose	≥ 100 - 125 mg/dL (impaired fasting glucose)
(may be normal in the early phases)	≥ 126 mg/dl Type 2 Diabetes Mellitus)

Note: NCEP ATP III = National Cholesterol Education Program Adult Treatment Panel III. Three or more indicators signal metabolic syndrome. Insulin levels are not commonly measured in a primary care setting.

DEFINING THE COMPONENTS OF THE METABOLIC SYNDROME

The metabolic syndrome has four recognized components. The relationships among these conditions—central obesity, hyperinsulinemia, dyslipidemia, and hypertension—have been the subject of much research in the past decade. The following sections review key elements of the research describing those relationships.

Table 2 Risk Levels Corresponding to Body Mass Index (BMI)

Height	Minimal Risk (BMI Under 25)	Moderate Risk (BMI 25–29.9) Overweight	High Risk (BMI 30 and Above) Obese
4'10"	118 lb or less	119–142 lb	143 lb or more
4'11"	123 or less	124–147	148 or more
5'	127 or less	128–152	153 or more
5'1"	131 or less	132–157	158 or more
5'2"	135 or less	136–163	164 or more
5'3"	140 or less	141–168	169 or more
5'4"	144 or less	145–173	174 or more
5'5"	149 or less	150–179	180 or more
5'6"	154 or less	155–185	186 or more
5'7"	158 or less	159–190	191 or more
5'8"	163 or less	164–196	197 or more
5'9"	168 or less	169–202	203 or more
5'10"	173 or less	174–208	209 or more
5'11"	178 or less	179–214	215 or more
6'	183 or less	184–220	221 or more
6'1"	188 or less	189–226	227 or more
6'2"	193 or less	194–232	233 or more
6'3"	199 or less	200–239	240 or more
6'4"	204 or less	205–245	246 or more

Note. From *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults* (Report No. 51S-2095), by the National Heart, Lung and Blood Institute, 1998, Washington, DC: Author.

Central Obesity

Prospective studies of adults and children have shown that central obesity increases the risk for development of type 2 diabetes and cardiovascular disease (Cigolini et al, 1996; Goran & Gower, 1998). Ample evidence suggests that this increased risk can be explained at least partly by the presence of central obesity along with the cluster of symptoms known as the metabolic syndrome (Lemieux et al., 2000). Disturbances in lipoprotein metabolism and insulin-glucose homeostasis and increased blood pressure have been repeatedly documented in individuals with central obesity (Juhaeri et al., 2002; Nieves et al., 2003).

Generalized obesity refers to increased weight in relation to an individual's height. Central obesity refers to excess adipose tissue residing primarily in the upper body within the abdominal region, whereas lower body obesity, or gynecoid obesity, refers to excess adipose tissue that resides primarily in the buttocks and thighs. Individuals with generalized obesity and an elevated BMI may or may not have a harmfully thick waist circumference that indicates central obesity. Further, relatively small individuals or individuals who are not overweight or obese may have an unusually large waist circumference that increases their risk For the metabolic syndrome. Therefore, practitioners must obtain a waist circumference measurement no matter what the patient's overall body habitus. In short, a tape measure may be one of the most effective tools for assessing a patient's risk for the metabolic syndrome and for the deleterious outcome of cardiovascular disease.

Generalized obesity can be determined by calculating the BMI and is defined as weight in kilograms divided by height in meters squared (kg/m^2). Individuals with a BMI equal to or greater than 25 are considered to be overweight and at risk for developing cardiovascular disease (see Table 3). Central obesity is thought to be a better indicator For the development of the metabolic syndrome and can easily be screened for by measuring an individual's waist circumference. Waist circumference that is greater than 102 cm (40 in.) in men and 88 cm (35 in.) in women is thought to considerably increase the risk for the development of the metabolic syndrome and subsequently type 2 diabetes and cardiovascular disease (see Table 4).

Hyperinsulinemia

Hyperinsulinemia, or insulin resistance, is associated with a constellation of risk factors leading to type 2 diabetes and cardiovascular disease (Bjorntorp, 2001; Nilsson et al., 2002). Prior to formal diagnosis of a pathological condition, individuals diagnosed with type 2 diabetes and cardiovascular disease have already experienced years of the harmful effects of the precursor metabolic syndrome (Alessi, Morange, & Juhan-

Vague, 2000; Juhan-Vague, Alessi, & Vague, 1991; Reaven, 1988). During this precursor metabolic syndrome, the groundwork for the significant vascular injuries and complications leading to type 2 diabetes and cardiovascular disease has already been laid (Alessi et al.; Juhan-Vague et al).

Early in the metabolic syndrome, the fasting glucose levels are within normal limits. This fact is due to a chronic state of hyperinsulinemia; that is, as long as the pancreas is able to produce sufficient insulin, glucose levels remain normal (Adami, Ravera, Marinari, Camerini, & Scopinaro, 2001; Anderson et al., 2001). Over time, the pancreas fails to maintain sufficient insulin, and glucose homeostasis is disrupted, resulting first in impaired glucose tolerance and then in hyperglycemia and overt type 2 diabetes (Reaven, 1988). Less is known regarding the exact role insulin resistance plays in cardiovascular risk; however, evidence suggests that there is a valid link between hyperinsulinemia and cardiovascular disease (Alessi et al., 1997). Hyperinsulinemia is a component of the metabolic syndrome, but insulin levels are nor commonly measured in a primary care setting. The norms for hyperinsulinemia have not been definitively established; however, evidence indicates that insulin levels higher than 17 uU/ml are more consistent with the development of the metabolic syndrome and subsequently type 2 diabetes and cardiovascular disease (Appel, Harrell, Davenport, & Hu, 2002; McMurray, Bauman, Harrell, Brown, & Bangdiwala, 2000; Monzillo & Hamdy, 2003).

Table 3 Generalized Obesity in Adults

Definition of Obesity in Adults, Based on Body Mass Index (BMI)	
BMI < 18.5	Underweight
BMI 18.5–24.9	Normal
BMI 25.0–29.9	Overweight
BMI 30.0–34.9	Class I Obesity
BMI 35.0–39.9	Class II Obesity
BMI > 40.0	Class III Extreme Obesity
BMI kg/m²	

Note. From *Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults* (Report No. 98-4083), by the National Heart, Lung, and Blood Institute, 1998, Washington, DC: Author.

Table 4 Waist Circumference Limits for Adults

Gender	Waist Circumference
Men	≥ 102 cm (40 in.)
Women	≥ 88 cm (35 in.)

Note. Exceptions for individuals shorter than 5 ft or individuals with a BMI greater than 35 kg/m² .

Although insulin levels are not commonly measured in primary care, there are several clinical markers that should be assessed as probable indicators of hyperinsulinemia. All clients should be assessed for acanthosis nigricans (AN). AN is characterized by hyperpigmentation, or velvety plaques of body folds (e.g., back of the neck, axillae, groin, elbows, or knees; Mukhtar, Cleverly, Voorhees, & McGrath, 2001). Community health screenings for AN have commonly focused on the back of the neck for identification of those individuals most likely to have hyperinsulinemia and to be at risk For the metabolic syndrome (Bent et al., 1998; Mukhtar et al.). Schools of the southwestern United States have used AN to successfully identify children and adolescents at risk for type 2 diabetes. The exact etiology of AN is not known; however, it is believed to be caused by hyperinsulinemia. Hyperinsulinemia causes the binding of insulin to insulin-like growth Factor receptors on keratinocytes and fibroblasts, with resultant hyperplasia of the skin (Crux & Hud, 1992).

Another potential marker of hyperinsulinemia in women is hirsutism. Hirsutism is an excessive growth of thick, dark hair in locations where hair growth is atypical for a woman and more common for a man (Goodheart, 2000). However, it is imperative to note that male pattern hair growth of familial hirsutisrn may be common in women of certain ethnic groups (e.g., those from the Mediterranean or Indian subcontinents). If a woman does not see her hair growth pattern as different from her Female relatives, then it is likely Familial. Likewise, White women with dark hair and darkly pigmented skin may be more likely to be hirsute than are fair-skinned or blond women. Hirsutism is caused by abnormally high levels of the androgens that stimulate hair follicles to produce excessive facial and body hair (Goodheart; Kauffman, Baker, Dimarino, Gimpel, & Castracane, 2002). Hyperandrogenism is often caused by abnormalities of either the ovaries or the adrenal glands. Polycystic ovary syndrome (PCOS) is a common etiology of hyperinsulinemia; researchers believe that the increased insulin

levels hyperstimulace the ovaries to produce surplus androgens (Homburg, 2002). Women with PCOS commonly possess a history of menstrual irregularities, dysmenorrhea, glucose intolerance, obesity, and/or alopecia on the crown of the scalp. Therefore, both AN and hirsurism may be helpful in the identification of clients who might possess hyperinsulinemia and would benefit from screening for the metabolic syndrome.

Dyslipidemia

Associations between central obesity and elevated triglycerides, as well as between central obesity and reduced levels of the high-density lipoprotein cholesterol (HDL-C), impose a risk profile for cardiovascular disease—related morbidity and mortality (Lemieux et al., 2000). Triglyceride levels greater than 150 mg/dl, total cholesterol levels greater than 200 mg/dl, or HDL-C levels less than 50 mg/dl in men and less than 40 mg/dl in women are considered to be risk factors for cardiovascular disease. For example, researchers have found that men who are both centrally obese and possess elevated triglycerides have an increased risk of coronary artery disease (Lemieux et al.). Lemieux and colleagues studied 185 healthy nonsmoking men to determine if waist circumference and triglyceride concentrations together could predict the other characteristics of the metabolic syndrome. Findings revealed that 80% of the men with central obesity and elevated triglycerides also met the diagnostic criteria for the metabolic syndrome defined by the National Cholesterol Educational Program (NCEP) Adult Treatment Panel (ATP) (Ford et al., 2002; Lemieux et al.). A second study assessed the presence of coronary artery disease among 287 unrelated male participants who underwent an angiographic study for rerrosrernal pain (Lemieux et al.). Participants who had at least one coronary artery lesion with more than 50% occlusion and participants without coronary artery lesions were Further compared by obtaining waist circumferences and fasting triglyceride levels. Findings revealed that the men with both central obesity and elevated triglycerides had significantly greater coronary artery disease than did men with low waist circumference and normal triglyceride levels (Lemieux et al.). These two studies added credibility to the theory that central obesity coupled with one or more other components of the metabolic syndrome increased the risk for cardiovascular disease.

Hypertension

It is uncertain how hypertension is caused within the metabolic syndrome; but preliminary studies indicate that the elevated blood pressure may be the result of abdominal adipose tissue stimulating the renin-angiotensin system. The enlarged abdominal adipocytes have also been found to excrete angiotensin (Bastard et al., 2000; Marchesi et al., 1999). However, it is certain that central obesity combined with hypertension does significantly increase the risk of cardiovascular disease. The larger the amount of abdominal adipocytes and the more elevated the blood pressure, the greater the risk For cardiovascular disease. According to the new Adult Treatment Panel III guidelines, blood pressure is considered elevated if the systolic pressure is greater than 130 mmHg or the diastolic pressure is greater than 85 mmHg (NCEP, 2001).

THE MECHANISM OF ACTION FOR THE METABOLIC SYNDROME

Investigators have shown that central obesity correlates with elevated plasma levels of plasminogen activator inhibitor 1 (PAI-1), which has been strongly associated with dysfibrinolysis within the fibrinolytic system, meaning that the balance between normal clot lysis and clot formation is disturbed (Alessi et al., 1997; Bastard et al., 1995; Brodsky, Malinowski, Golightly, Jesty, & Goligorsky, 2002; Vague, Vague, & Cloix, 1971). It is this disturbance in clotting that has led investigators to focus on the plasma levels of PAI-1, which, if abnormally high, lead to both ruptured plaque and thrombotic occlusion. Ruptured plaque is considered an important mechanism in the development of a myocardial infarction (Brodsky et al.; Falk, 1992). Further, elevated plasma PAI-1 levels have been shown to predict infarction and stroke (Juhan-Vague & Alessi, 1996; Juhan-Vague, Morange, & Alessi, 1999).

Hyperinsulinemia has been directly associated with increased levels of plasma PAI-1 (Alessi et al., 1997). The exact mechanism by which elevated insulin levels stimulate increased plasma PAI-1 levels is still under investigation (Bastard et al., 2000). It is known, however, that the increased insulin levels over time increase the risk for developing type 2 diabetes, and it is also known that individuals with central obesity are at greater risk than those individuals with generalized obesity (Koistinen et al., 2000). Investigators are continuing to examine

the role of increased plasma PM-1 levels and elevated triglycerides and cholesterol. In the third National Health and Nutrition Examination Survey (NHANES III), researchers found that central obesity correlated with elevations in blood pressure and insulin levels, elevated plasma PAI-1 levels, and harmfully low HDL-C levels (Ford et al., 2002). The NHANES III also revealed that there were more women and minorities who possess central obesity and the metabolic syndrome. The NHANES III is of particular importance, as it is believed to contain a cross-sectional sample that is representative of the actual population of the United States.

TAKING THE MEASUREMENTS

The determination of central obesity is based on the location of adipose tissue in relation to anatomical abdominal structures. The fascial plane divides the boundaries between the two adipose layers made up of the abdominal wall muscles and the deep fascia of the paraspinal muscles (Ferland et al., 1989). The superficial subcutaneous adipose tissue lies above the fascial plane, and the visceral adipose tissue resides below the front plane.

There are three accepted techniques for measuring central obesity: imaging, waist circumference, and skin-fold thickness. Imaging techniques that measure adipose tissue provide the most direct and accurate measurements. The gold standards are magnetic resonance imaging, dual-energy X-ray absorptiometry, and computed tomography scans for direct determination of the degree of superficial subcutaneous versus deep visceral deposits of abdominal adipose tissue (Daniels, Khoury, & Morrison, 2000). However, these tests are expensive and time-consuming and expose the patient to radiation. Such limitations have led to an examination of the efficacy of indirect measurements that are comparable yet simpler and less expensive (Couillard et al., 2000; Daniels, Morrison, Sprecher, Khoury, & Kimball, 1999; Despres et al., 2000; Gower, Nagy, Goran, Toth, & Poehlman, 1998).

Investigators have shown that for adults, waist circumference is the best indirect measurement of fat distribution; when compared to studies using imaging techniques, waist circumference was the next most reliable method and was least affected by gender, race, or overall level of adiposity (Daniels et al., 2000; Maffeis, Grezzani, Pietrobelli, Provera, & Tato, 2001). The NHLBI (1998) has recommended waist circumference as a comparable indirect measurement for central obesity in patients for whom the direct methods are not clinically feasible.

A tape measure made by the Country Technology corporation, the Gulick II tape measure, can be used ***as*** an exact mea sure of waist circumference (Country Technology, 1999). The Gulick II is commonly used in clinical research studies, due to its no-stretch feature and the tensioning device attached to the rape. This device provides a known amount of tension during measurement. A stainless steel compression spring is used so that it is impossible to overcompress. The rape is read when wrapped around the participant at the "zero line." However, if the provider follows the NHLBI guidelines, an ordinary tape measure may be as useful for the general clinical screening that occurs in a primary care office. The measurement is taken with the patient standing, and waist circumference is measured From the uppermost lateral border of the iliac crest. People 18 years of age and older are considered to have central obesity if waist circumference exceeds 102 cm (40 in.) for men and 88 cm (35 in.) for women (Djousse, Rothman, Cupples, Arnett, & Ellison, 2003; NHLBI, 1998). According to the NHLBI, waist circumference cut points can be standardized across all adult ethnic populations, except for individuals shorter than 5 ft or individuals with a BMI greater than 35 kg/m^2. In these cases, BMI may be a more effective method of screening For cardiovascular risk (Djousse et al.).

FACTORS THAT CONTRIBUTE TO CENTRAL OBESITY AND THE METABOLIC SYNDROME

Lifstyle Factors

The most important factor leading to the development of central obesity is a lifestyle in which caloric intake exceeds physical activity, leading to both generalized and central obesity. Several studies have examined the association between smoking and central obesity. Although heavy smokers may have a normal BMI, they may also have larger waist circumferences than do nonsmokers, placing them at additional risk for development of the metabolic syndrome (den Tonkelaar, Seidel], van Noord, & Baanders—van Halewijn, 1990).

Familial Factors

A positive family history for either type 2 diabetes or cardiovascular disease coupled with obesity, including central obesity, significantly increases the risk for developing the metabolic syndrome (Dwyer et al., 1998; van Lenthe, van Mechelen, Kemper, & Post, 1998). Ford and colleagues (2002) examined data from the NHANES III to investigate the prevalence of the metabolic syndrome in the United States. Their study revealed a high prevalence of the metabolic syndrome among women and especially among minority women, including African Americans and Hispanics (Ford et al.). Further, it has been found that individuals whose heritage includes members of ethnic groups who generally have smaller body frames, such as Asians, are at an increased risk of developing central obesity when they do not appear to have generalized obesity (Harris, Stevens, Thomas, Schreiner, & Folsom, 2000; Kauffman et al., 2002). Likewise, elderly people may not manifest generalized obesity as they age but instead develop central obesity (Planas et al., 2001). Therefore, measuring central obesity may be crucial in determining risk for clients of normal weight.

Table 5 Characteristic Abnormalities of Insulin Resistance

Plasma Glucose:	**Abnormal Glucose Findings:**
Fasting Glucose:	**Impaired Fasting Glucose**
	110 - 125 mg/dl
	Type 2 Diabetes
	> 126 mg/dl
120-min post-glucose challenge (75 g)	**Impaired Glucose Tolerance**
	140 - 200 mg/dl
120-min post-glucose challenge (75 g)	> 200 mg/dl

Note: Diagnostic criteria from "Screening for type 2 diabetes" by the American Diabetes Association, 2004, *Diabetes Care, 27*(Suppl.) 1:S11-4.

Environmental Factors

A low socioeconomic status and lack of insurance or poor access to health care are risk factors for developing central obesity and the metabolic syndrome (Benjamin-Garner et al., 2002; Diez Roux, Jacobs, & Kiefe, 2002). Persons with low economic and educational backgrounds tend to consume diets higher in fat and calories and rend to exercise less than other groups (Appel, Harrell, & Deng, 2002; Carter-Edwards, Jackson, Runaldue, & Sverkey, 2002). Further, persons without access to preventive health care may not be aware of their risk for the development of hypertension, type 2 diabetes, or cardiovascular disease and subsequently may not address their individual risk (Carter-Edwards, Skelly, Cagel & Appel, 2004; Cagel, Appel, Skelly & Carter-Edwards, 2002).

IMPLICATIONS FOR PRIMARY CARE PROVIDERS

Physical examination of the patient should assess the degree and distribution of body Fat, patterns of hair growth, uneven skin coloration, nutritional status, exercise history, and any signs and symptoms of secondary causes of obesity. Less than 1% of all obese clients have an identifiable secondary cause For the obesity; but hypothyroidism and Cushing's syndrome should be evaluated as potential causes For recent unexplained weight gain.

Both BMI and central obesity measures should be taken on all adult clients. Particular attention should be given to individuals who have upper body obesity or central obesity, because Fat distributed around the waist is considered a greater health risk than fat distributed around the flank, thighs, and buttocks. Women of African American and Hispanic descent are at greater risk for developing central obesity than are women from other

ethnic groups (Ford et al., 2002). All obese clients should be encouraged to participate in a diet and exercise program that will facilitate weight loss. Such participation is difficult for most clients, and the success rate For these types of programs is very low. Prescription medications or surgery should be options for only the morbidly obese client who has not had *success* losing weight with other behavior modification programs.

Practitioners should use measurement of waist circumference, a fasting glucose level, lipids, and blood pressure to assess all obese clients for components of the metabolic syndrome. When components of the syndrome are identified, a 2-hr Fasting oral 75-g glucose tolerance test is an appropriate evaluation of glucose metabolism (see Table 5), even with a previously normal morning fasting glucose, as a challenge test is more definitive (American College of Endocrinology, 2003; American Diabetes Association, 2004). Clients with symptoms severe enough to warrant a diagnosis of impaired glucose tolerance, type 2 diabetes, elevated triglycerides, low HDL-C, or hypertension should be treated with appropriate pharmacological therapy. Pharmacological agents that improve the profile of the metabolic syndrome include vitamin supplements containing vitamin E (1,200 IU; Devaraj, Chan, & Jialal, 2002), angiotensin inhibitors for the treatment of hypertension, cholesterol-reducing agents, antiplatelers such as a daily aspirin (81-325 mg), and oral hypoglycemic agents (Glucophage and/or Actos) For the treatment of impaired glucose tolerance and ultimately type 2 diabetes (Knowler et al., 2002). Research indicates that the thiazolidinediones, or glitazones, are beneficial for the metabolic syndrome, as they reduce levels of PAI-1 and triglycerides and raise HDL-C.

CONCLUSION

Growing evidence suggests that central obesity plays a more significant role in the development of the metabolic syndrome than does generalized obesity. In the past, primary care providers have focused primarily on body mass and overall weight indexes to measure obesity in relation to the development of type 2 diabetes and cardiovascular disease. Current standards recommend the measurement of central obesity in the overall treatment of adults, especially among women and minorities. Although it is not feasible to measure either insulin levels or plasma PA1-1 levels in primary care, measurement of waist circumference, BMI, lipids, blood pressure, and fasting glucose as well as being alert for presence of AN and/or hirsutism are a feasible first step in formulating an accurate patient risk profile. It is essential for primary care providers to remember that they will be the first to see clients at risk for and with the metabolic syndrome. Early interventions related to diet and lifestyle are needed to stop this dual epidemic of obesity and type 2 diabetes. When clients manifest the metabolic syndrome, primary care providers should be ready to use a formal glucose tolerance test to screen For type 2 diabetes. Such testing will Facilitate early interventions and thus minimize the deleterious effects of the syndrome.

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